

ON SENSORIMOTOR PALSIES OF THE MUSCULATURE
OF THE FACE, WITH REMARKS ON THE OCULAR
PALSIES OF THE EARLY STAGES OF TABES.¹

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About one year ago I reported to this society a case of disease of the base of the brain with implication of the fifth nerve, and remarked on that occasion, when speaking about the motility of the face, the following: "The examiner's attention is first arrested by the patient's facial expression. The left side of the face seems contracted; the left-sided temporal and masseter muscles appear atrophied. The left eyelid is drooping. The degree of contraction of the muscles of the left side of the face and the width of the left palpebral fissure are by no means constant; they vary greatly from day to day, and even during one examination. These variations are greater than would correspond to mere physiological changes of the physiognomy, and are confined to the diseased side. For instance, the lid that was drooping at one time is unduly elevated at another time, exposing a part of the upper margin of the sclera. At first it appears as if the right-sided facial nerve were paralyzed, because the folds around the eye and angle of mouth of the left side are much deeper. However, when the patient is put through the usual tests to determine the state of the facial nerve, the right facial nerve is found to be absolutely normal, while in the distribution of the left a phenomenon is noticed, that can best be compared to the ataxia and over-innervation commonly observed in the extremities of tabetic patients. The patient performs all motor functions with the left facial nerve, closing and opening the eyes, showing the teeth, etc., with unnecessary force, with an expenditure of energy that is extravagant compared to the effect desired. At other times these same

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movements are slow, awkward and peculiarly jerky and interrupted."

I have been impressed with the diagnostic significance of the facial expression, as no doubt a great number of us have been. We all have probably evolved for ourselves some clinical rules which frequently enable us to read the disease from the expression of the face. I need only remind you of the Parkinsonian face, of the facial expression of a general paretic or a patient afflicted with bulbar paralysis. Among the tabetic patients a characteristic facial expression is also frequently observed, and I have attempted to analyze this phenomenon since I reported to you the above case.

During these studies good fortune brought another case under my observation which presented many points particularly serviceable in elucidating part of the question to which I propose to invite your discussion to-night. The suggestions received from the first case proved quite helpful in the analysis of the second, to be reported presently, and from this analysis the following points crystallize out for further more thorough investigation:

1. What, if any, is the effect of disease of the fifth nerve upon the motility of the face?
2. Which, if any, function of the fifth nerve is it in particular that is indispensable for perfect motility of the face?
3. The fifth nerve carrying deep sensibility to the ocular musculature as well as to the facial, what—if any—is the effect of disease of the fifth nerve upon the motility of the former?
4. How are we to clinically determine the state of deep sensibility of the facial and ocular musculature?
5. Are not some of the ocular palsies observed in the early stages of tabes sensorimotor paralyses and not primarily motor?

For the solution of these questions the following methods were employed:

1. Study of a case with a rather fortunate grouping of symptoms.
2. Study of the physiological, clinical and anatomical literature at my disposal.
3. Analysis of twenty-two cases of tabes, at present under

my observation, with regard to the relation between disordered function of the facial and oculomotor nerves² and disease of the fifth nerve.

In looking over the histories of other cases of tabes observed during the last six years, I found them insufficient in the particular points under discussion, and as the findings could not be verified now, they were unreliable for the present investigation.

The case referred to is the following:

I. B., forty-two years old, a Roumanian musician, was first seen by me when he was shown in a medical society of this city as a case of *tic douloureux*, two years previous to his admission to the Montefiore Home.

At that time he complained of attacks of severe pain in the left side of the face and in the areas of distribution of the trigeminal nerve; and of the sensation of a heavy helmet on his head.

On October 6, 1898, the patient entered the Montefiore Home and the history obtained after admission to the hospital is as follows:

Born of healthy stock and temperate in his habits, he was well up to his twentieth year. When he was five years of age a hot drink "burned" his throat and the result of this is found to-day in an extensive synechial adhesion between the posterior surface of the soft palate and the anterior surface of the pharyngeal wall. The patient married at the age of eighteen, became father to five healthy children, one of whom died at an early age. His wife never had a miscarriage. He believes that he had a venereal sore when twenty-one years of age. This disease was treated internally and externally during four months and he has not had any further manifestations of the infection since that time. Thirteen years ago an attack of pain and inflammation (probably glaucomatous) destroyed the vision of his left eye.

The present disease began from unknown causes three years ago with attacks of excruciating pain in the face and head, preceded and followed by a sensation of a heavy helmet upon the head and face (*Hutchinsonian mask*). One and a half years ago vision of the right eye began to fail and patient is at present absolutely amaurotic. He has had, and has now, paresthesias here and there in the extremities, particularly in

² The term *oculomotor nerves* is used here and in other places of this paper in the sense of the general nerve-supply of the external eye-muscles.

the upper ones. No vesical disturbances were noticed at any time. Bowels are costive since onset of the disease and sexual appetite is considerably diminished. During the present illness there was no vomiting. He is at present moaning and groaning day and night, and claims to have unbearable pain and paresthesias in the head and face, particularly on the left side.

He is well built, poorly nourished, and shows few somatic signs of degeneracy. Station and gait are uncertain and ataxic, particularly the station.

The most remarkable symptom is the patient's facial expression. When he is quiet, one would think that there were a paralysis of the lower two branches of the left facial nerve. When he is made to laugh or cry, or when he does so in response to autochthonous psychical stimulation, only the upper branches of the left and the lower branches of the right facial nerve go into play. This gives to the face an indescribably contorted expression. These movements show, moreover, a considerable over-innervation; the contractions of the left frontalis and orbicularis oculi and of the right buccal musculature are executed with a good deal of force and vigor, quite disproportionate and not alike on all occasions. Apparently as a result of this phenomenon the skin around the left ocular and right oral aperture is thrown into deep folds while the rest of the face continues in a smooth, masklike expression. This expression is vastly different from the Parkinsonian facial immobility; the stiffness of the latter is replaced here by an atonic relaxation of the facial musculature. The left palpebral fissure is of varying width; at a given moment the fissure is smaller than normal; at another the upper lid is unduly elevated, exposing the upper scleral margin.

The head is in general tender to percussion, perhaps a trifle more so in the occipital region. The head is moved freely in all directions, but passive excursions of the head appear to be painful, to judge from the change in the facial expression. On such an occasion the lower branches of left facial, so far apparently paralyzed, bring the muscles about the angle of mouth into vigorous contraction and partly equalize the mentioned facial asymmetry.

Sense of smell and sense of taste are considerably impaired on both sides.

The patient is completely amaurotic; the ophthalmoscope reveals a complete bilateral optic atrophy. The ocular axes are divergent. Left pupil is key-hole shaped and obstructed by a grayish mass. Right pupil is well shaped and reacts to light. When the patient is made to look at the point of his nose and then into the distance the right pupil dilates in the first instance

and contracts in the second. The excursions of the right eyeball are normal. The muscles of left eyeball seem at first parietic, the excursions being very much less prompt and below the physiological limits. In lateral positions nystagmiform twitchings are observed. When patient is made to repeat frequently the task of bringing the left eye into different positions the promptness and vigor of the contractions improve gradually and are finally fairly good, though not as good as on the other side, but decidedly *better* than they were at first. The nystagmiform tremor of the eye muscles is also considerably lessened after such exercise.

On a later occasion the same exercise has to be repeated; the improvement in the motility of the eye muscles of the left eye not being permanent. The extent of the limitation is equally unlike on all occasions as are also the contortions of the face and the width of the left palpebral fissure.

To all appearances not one of the external eye-muscles of left side is found actually paralyzed, but all the movements of this eye are unequally balanced and co-ordinated; exaggerated or diminished on different occasions and improve considerably under exercise.

Active innervation of facial musculature is similarly undisturbed in the common sense of the term; all muscles respond to intentional innervation, but with varying force on different occasions, particularly on left side. The temporal and masseter muscles of left side are atrophied, and when the inferior maxilla is moved downward it deviates toward the right side.

Soft palate is attached to pharynx, is immovable, and this impairs considerably the swallowing and speech of patient. The speech is, however, not only nasal but otherwise dysarthric.

Tongue is protruded straight, freely movable in all directions, of good color and shows no tremor or atrophies. The patient lost all his teeth without pain during the present illness.

Extensive disturbances of the sensibility in the distribution of the trigeminal nerves, particularly of the left, are found, and comprise all qualities of sensibility including the deep sensibility. I shall speak later in regard to the method of ascertaining the latter.

The upper extremities show good motor and co-ordinating power and the deep reflexes are present. Undeniable hypotonia exists.

No paralyses or atrophies are found on the trunk. Scattered areas of anesthesia, analgesia and delayed conduction are found. Skin reflexes are present.

On lower extremities slight ataxia and marked hypotonia

are observed; knee- and Achilles-jerks are absent; plantar reflexes are lively. Considerable disturbances of tactile, pain- and temperature-sense exist on the feet and lower third of the legs; deep sensibility apparently intact. Vegetative organs normal.

The final diagnosis of the case reported, tabes or tumor on or near the cerebellum is somewhat doubtful. All evidence points rather towards the diagnosis of tabes. There is unmistakably extensive disease of the fifth nerve, particularly on the left side, and the symptoms dwelled upon at length are clearly dependent upon the trigeminal disease. To recapitulate these symptoms briefly, we could say that there is motor impairment of the facial and ocular musculature, particularly of the left side. The motor defects are of varying intensity and extensity, and are defects of the co-ordination rather than of the motor power proper, as is shown by the fact that the extent of the paralysis varies on different occasions; and particularly concerning the eye-muscles by the fact that exercise overcomes a part of it. Disease of the fifth nerve is evidenced by the disordered sensibility in the distribution of the fifth nerve, particularly on the left side, by the atrophies of the temporal and masseter muscles of the same side, and finally by the loss of the teeth. The tactile, pain and temperature anesthetics were mostly marked in the distribution of the first branches of the trigeminal and were varying; the deep sensibility, however, was permanently and considerably injured.

Before entering upon the further discussion and before attempting to answer the questions formulated at the outset, a physiological preface and short review of the literature seems apropos.

For various reasons it is helpful and instructive to establish an analogy between the cranial and spinal nerves, and further between the cranial and spinal anterior and posterior roots. This analogy may not be anatomically exact, but is quite probable from the embryological and physiological standpoint. So we may compare the trigeminal nerve to a posterior root, and look upon its motor portion, the facial, the oculomotor and trochlearis, as the anterior partner. Excepting, perhaps, the vegetative functions, particularly the motility of the heart where

an automatic apparatus is believed to regulate the motor variations of this organ, it is gradually developing into an established fact for the cerebrospinal functions, that all centrifugal nerve impulses are not autochthonous and independent. In other words, the belief is forced upon us that all centrifugal nerve functions—motility, secretion and trophic influences—are conditioned and regulated by centripetal or sensory impulses. This is well supported by experimental and pathological experience. We all know that section of the posterior roots leads not only to disturbance of the sensibility, but of motility as well. The latter is, of course, not a paralysis in the current sense of the term; the motor functions are altered after section of the posterior roots in kind only; the movements lose their purposefulness and proper adjustment. So far the only tenable explanation of ataxia seems the Leyden-Goldscheider theory, and the ataxia without sensory disturbances called for by Goldscheider is yet to be shown.

Marinesco has furthermore proven by anatomical investigation that abolition of the nerve impulses traveling constantly along centripetal pathways finally derange the structure of the motor apparatuses with which these pathways are anatomically and physiologically connected. Oppenheim and Schlesinger finally succeeded in finding the intermediate centripetal pathways along which the ascending degeneration is supposed to travel.

The influence of sensibility upon the motility is discussed with fair interest in physiological literature. In the neurological text-books and pamphlets this subject is treated rather briefly, and particularly the question concerning the changes of the facial and ocular motility as influenced by sensibility, most text-books pass tacitly.

Exner defines sensomobility as the ability to perform motor functions in so far as they are guided, influenced or regulated by centripetal nerve impressions. Disturbances of the sensibility, according to this author, lead to various alterations of the motility, depending upon the extent and kind of the former as well as the latter. These motor disturbances are produced by the suppression of one or more of the sensory regulations. As

yet there are no distinct differences between the various forms of sensorimotor palsies.

It was known already to Bell and Magendie that the state of sensibility of the face greatly influenced the motility. Experiments led these authors to the conclusion that cutting the fifth or seventh nerve caused the same result, *i. e.*, inability of the animal to grasp its food, but for different reasons: in the first instance owing to the disordered sensation, in the second to the disordered motion. Here we find explanation for the fact that Bell frequently expressed the opinion that the fifth nerve is a motor nerve in some respects. Disturbances of the sensibility of the tongue do not often demonstrably cripple the mobility. The fact that the lingual as well as facial musculature is most of the time bilaterally innervated accounts for the rarity of clearly demonstrable motor disorder when the sensibility is interfered with. In functions that are unilaterally represented, as for instance the function of speech, disturbances are frequently seen. Dysarthria and thickness of speech is frequently seen when the sensibility of one-half of the tongue has suffered. A good illustration of the interdependence between sensation and motion is the experiment of Exner in which he succeeded in producing a paralysis of the vocal cords after section of the laryngeus superior.

Landois, when speaking about this subject, states that sometimes section of sensory fibers is followed by total abolition of the motor function.

In Baker and Harris' "Handbook of Physiology," ed. 1885, the following is found: "In relation to muscular movements the branches of the greater portion of the fifth nerve exercise a manifold influence on the movements of the muscles of the head and face and other parts in which they are distributed. They do so in the first place by providing the muscles themselves with that sensibility without which the mind, being unconscious of their position and state, cannot voluntarily exercise them. It is probably for conferring this sensibility on the muscles that the branches of the fifth communicate so frequently with those of the facial and hypoglossal and the nerves of the muscles of the eye."

On the other hand, Schiff feels justified, from his experi-

ments, in expressing his view to the effect that after section of the fifth nerve on one side, the movements of the face are equally well executed on both sides, and that only as a matter of course the animals, being robbed of the sensation in one-half of the face, will contract these muscles less frequently. He therefore thinks it erroneous to state that cutting the fifth nerve interferes with the motility of face or eyes.

This chapter is apparently beginning to absorb more neurological interest to judge from the controversy between Korniloff and Bickel in the XII and XIII volumes of the *Deutsche Zeitschrift für Nervenheilkunde*. Korniloff exhibited at the last meeting of the south German neurologists and psychiatrists a series of animals with severed posterior roots. In all of them there was more or less extensive disorder of the motility, amounting in some to apparent total paralysis. Korniloff further endeavors to find the kind of sensibility that may be incriminated; but the facts so far collected do not permit a final conclusion. Bickel opposes these deductions and claims that an undisturbed sensibility is not indispensable for the soundness of the motility. It seems to me that Bickel is taking the word paralysis in the usual sense, meaning actual motor palsy, and that he would be willing to admit that sensory disorder changes the motility where co-ordination and reflex motion are concerned.

Manifestly the sensorimotor inter-relation has commanded considerable physiological thought since the attention was first drawn to it by Bell and Magendie, and it is evident that all physiologists, excepting Schiff, agree that this interdependence is a considerable one.

As stated already this subject has received less thorough consideration at the hands of the neurologists. Gowers believes that "the movements of the head, face, tongue and eyes escape the characteristic derangement of tabes," although he states, when speaking about the symptomatology of disease of the fifth nerve, that "the muscles of the face are insensitive but not weakened, although the movements of the face have been observed to be a little slower than normally, apparently from defective sensation."

In Dana's text-book the relation between sensation and motion in the face is not discussed. The author remarks, however, that "a slight drooping of one or both lids is not infrequent in the early stages of tabes. It begins early and progresses slightly up to the later stages of the disease. It is due to a paralysis of the sympathetic nerve fibers of the lid."

Dercum's text-book contains from the pen of Peterson the statement that the muscles of the trunk are also ataxic, but the inco-ordination does not affect the movements of the face, tongue, eyes or head. In the same volume Herter does not discuss the influence of disease of the fifth nerve upon the motility of the face.

Rosenthal's "Treatise on Nervous Diseases," translated by Putzel, contains a few very suggestive lines. He states: "The trigeminal nerve is sometimes implicated in ataxia. In a case observed by Duchenne, double paralysis of the fifth pair of nerves occurred with paralysis of the left motor oculi communis. In four of my cases paralysis of the trigeminal and motor oculi communis occurred twice, paralysis of the latter nerve and of the facial and trigeminal developed once. * * * The facial nerve is rarely involved, but certain muscles of the face manifest diminished tonus."

In the newer text-books of Oppenheim and Schultze the sensorimotor inter-relation is very briefly alluded to.

The facial nerve is unanimously reported to be very rarely affected in tabes, and it is perhaps probable that in the cases in which such affection was observed it was not a genuine facial palsy, but a disordered facial motility of the type discussed.

The question is of considerable neurological interest in regard to the pathogenesis of tabes. We frequently hear the objection raised when we attempt to look upon tabes as a disease of the sensory neurons, that the facial and oculomotor nerves are not sensory nerves, and both are involved in the tabetic process, the latter vastly more frequently than the former.

The ocular palsies are according to all authors very frequent and very early symptoms of tabes. With equal uniformity these ocular palsies are characterized as dissociated, slight and transient. All these characteristics are not fully compati-

ble with some of our cardinal conceptions about disease of the motor neuron, and the nature of these ocular palsies is therefore still *sub judice*.

Not much light has as yet been thrown on this question by histological and anatomical investigation. Negative post-mortem findings are just as often reported as degenerative changes in the nucleus, peripheral fibers, or in the muscles. de Schweinitz writes in Dercum's handbook concerning this question thus: "We are uncertain as to the exact cause of these palsies, some being explained as nuclear, others as the result of a descending neuritis, and still others as due to a chronic ependymitis. One of the characteristics of tabetic paralysis is its transitory nature in many instances, and by this peculiarity it may often be differentiated from the lesions of the nerve trunks themselves."

Thus the matter stands at present—quite remote from a satisfactory final solution and hence the justification for the attempt to view the question from some uniform standpoint.

All that seems so far established about the tabetic ocular palsies is the following:

1. They are frequently sudden in onset, of short duration, and disappear just as suddenly without treatment; or they are of longer duration and then disappear mostly under specific medication, or they are permanent.
2. They are frequently dissociated (paralysis of the levator or rectus internus alone), or the paralysis affects all muscles or muscle groups supplied by a given nerve.
3. The extent of the paralysis is sometimes, as I have observed in the case reported and in two others included in the appended table, varying in short intervals and frequently diminished after exercise.
4. Their anatomical substratum is not one and the same; negative findings are just as often reported as lesions of the respective nuclei, nerve trunks or muscles.

Theoretically, there are only two possibilities: first, these ocular palsies are either pathological occurrences of another nature than the general disease they are implanted upon; they share with the general disease the common etiology only, *i. e.* the syphilitic infection, and are pathologically primary or sec-

ondary nerve inflammations in distinction from the pathological lesion of the general disease—a primary degeneration of the sensory neuron. Or, secondly, these ocular palsies are a part of the general disease, *i. e.*, a primary degeneration of the sensory neuron. The degeneration aided by so far unknown circumstances affects in some cases the sensory neuron of the cephalic extremity, and leads to first functional and later structural changes of the corresponding motor neuron—in this instance the oculomotor nuclei, nerve trunks or muscles.

It seems that both mentioned types of ocular palsies are met with in tabes and that the fact of their not being sufficiently kept asunder is obstructing the way to a clearer conception of their pathogenetic relationship to the general disease. The first type is oftener seen in those cases that are rather forms of multiple cerebrospinal syphilis of the tabetic type, and the second in the class of cases that lean more towards the picture of a genuine tabes. In the first instance they may therefore be looked upon as syphilitic ocular palsies occurring in a case of multiple cerebrospinal syphilis of the tabetic type; and in the second instance they are essentially the same phenomena as the motor phenomena in the extremities of tabetics.

It is for the second type of ocular palsies that the view of a primary sensory origin seems to me justified.

These palsies are in their earlier stages simply due to the loss of tonus which is mediated through disease of the sensory neuron, and later on when owing to the long duration or the severity of the process in the sensory neuron the motor neuron has undergone secondary changes, the palsies are lasting, more extensive and their immediate cause found anatomically in degeneration of the nucleus, nerve trunk or muscle. This view is not only quite compatible with the clinical and anatomical characteristics of this type of palsies outlined above, but receives some further support from the fact that it permits a uniform pathogenetic conception of the general disease, and is finally gaining probability from the results of the analysis of the twenty-two cases appended.

The analysis of these cases was undertaken with the aim to ascertain the relation between defective function of the facial and ocular musculature and disease of the fifth nerve. I am

indebted to Dr. Abrahamson for having kindly tabulated for me the results and made the examinations in the following way:

Every patient was examined as to the previous or present existence of trigeminal or oculomotor symptoms. The previous existence of oculomotor symptoms was assumed from a history of double vision or squint; and previous trigeminal symptoms from paresthesias or pain in the face, from masticatory spasms, eye inflammations or loss of teeth. In the examination of the present condition we proceeded in the following way: 1, examination of the ocular excursions; 2, examination of the trigeminal nerve; 3, examination of deep sensibility of face and eyes. The deep sensibility of the facial musculature was ascertained by means of the faradic current, and the patient was interrogated as to his knowledge of the degree of a given contraction. These findings are frequently unsatisfactory. I derived more satisfaction from the procedure of ascertaining the deep sensibility of the eyelids. The most satisfactory results were obtained from two patients, the one reported in this paper and another included in the table, both of whom are totally amaurotic.

When the lid is carefully and gently lifted up by the eyelashes in the external canthus, the patient either distinctly states that the lid is moved upward or downward when the deep sensibility is intact; otherwise he is just as misinformed about the position of his eyelid and looses it somewhere on the eye, as the tabetic patient looses his limbs in the bed. With unimpaired vision the test is less accurate, but still reliable if the examination is made with caution and patience. Under such circumstances the patient is made to look fixedly at an object in the center of the visual field, while he is directed to answer quickly, without making searching movements with the lid after it was lifted or lowered, what the examiner did with it.

In order to determine to what extent the defect of the deep sensibility of the lids is caused by the concomitant amaurosis, the same examinations were made with a large number of other patients. In this type of cases the passive excursions of the lids are acutely and promptly perceived, but it must be stated that in amaurotic persons the eyelids are frequently found

drooping, although the levator palpebræ superioris is otherwise quite intact. In this instance the semi-ptosis is also an expression of a primary sensory defect—the disease of the optic nerve. The endeavor was further made in case evidence of disease of the ocular muscles was obtained, to ascertain to what extent exercise would overcome the existing defect.

Finally attention was paid during the examination to the state of the facial and trigeminal nerves in their lower branches. The detailed results of this examination are embodied in the appended tables and I shall give here only a condensed synopsis of same.

1. Out of the 22 cases examined, 10 give distinct evidences of a previous venereal infection; in 4 cases an infection may be assumed from the accompanying symptoms, and in 8 no reliable information could be gotten.

2. Eight out of the 22 cases give a history of derangement of the ocular musculature previous to the date of the examination, and in 14 no such history was obtainable. Thus it would appear that ocular palsies occur in 36.4 per cent. of the cases of tabes.

3. Two of the 8 cases show a distinct history of previous syphilitic infection; in 2 a venereal infection is probable, and in 4 it is apparently absent.

4. All 8 cases showed evidence of disease of the fifth nerve.

5. Amongst the 14 cases that were apparently free from disease of the muscular apparatus of the eye, 8 had a venereal infection, 4 denied the same, and in 2 it was probable.

6. In 6 out of the 14 cases a history of derangement of the fifth nerve was obtainable. These are cases 1, 4, 10, 12, 15, 17, and it is interesting to note that in cases 1 and 4, optic atrophy was the earliest symptom, so that an eventual disorder of the ocular musculature may have escaped being noticed by the patient. This is the more probable, as both these cases show at present both disease of the muscular apparatus of the eye and of the fifth nerve. In the other 4 cases the only evidence of previous disease of the fifth nerve was surmised from the statement of a painless loss of teeth, *a priori* not very likely to be thought of as conditioning disorder of the motility of the ocular musculature.

7. Six out of the 22 cases show at present evidence of disease of the ocular muscles. Two cases give a distinct history of syphilis, in one the infection is probable, and in three apparently absent.

8. In all these 6 cases there is evidence of extensive disease of the fifth nerve, and in 5 the deep sensibility of the eyelids was found markedly disturbed.

9. Sixteen cases show no disorder of the eye-muscles at present, and in 9 of them disease of the fifth nerve was demonstrated, but only in 3 the deep sensibility of the eyelids was defective.

10. It is very remarkable that 15 of the 22 cases show more or less marked ptosis, or better said, more or less considerable narrowing of the palpebral fissure.

11. In 6 out of the 22 cases derangement of the facial musculature was found, and in all these corresponding derangement of the fifth nerve.

The reasons stated above and the results of the table corroborate the belief in the possibility that some of the ocular palsies of tabes may not be purely motor, but sensorimotor paralyzes, conditioned through primary disease of the sensory neuron and not due to primary disease of the motor neuron.

Further support is lent to this conception by the results of experimental and anatomical investigations made by Marinresco, Oppenheim, Schlesinger and Grabover, who traced disease of motor cells or fibers to primary disease of the corresponding sensory neurons.

Now we may attempt to answer the questions formulated at the outset:

1. Disease of the fifth nerve interferes with the motility of the face.

2. As yet no function of the trigeminal in particular can be ascertained which could justly be supposed to be the cause of the impaired motility; the kinesthetic sensibility when cautiously and patiently examined was found more frequently and more extensively disordered than the other functions.

3. The effect of disease of the fifth nerve upon the motility of the eyes seems to be a similar one.

4. All the means at present at our command for examining

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 1—Denied. Two miscarriages.	Apparently not.	Paresthesias over forehead, both eyes and cheeks. Loss of teeth—painless.	Convergent strabismus. Weakness of both external recti, especially right, both superior recti, marked ptosis on both eyes, especially left.	Sensibility: tactile: spots of anesthesia on forehead, hypesthesia on tip of nose. Pain: large areas of analgesia especially left face; also over the right face, excepting portions of cheeks and forehead where also some hyperalgesia. Temperature: normal. Deep: disturbed in both eye-lids.	Normal.	The early optic atrophy probably prevented the recognition and perception of ocular palsies by the patient, even if they had existed at the earlier stages of the disease. The convergent strabismus is extreme, but after exercise patient succeeds in bringing the eye-balls fairly well out to right and left side—after exercise the paralysis disappears almost entirely.

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 2—Denied.		Paresthesias over forehead, eyes and cheeks. With the onset of disease had without assignable cause a conjunctivitis—not severe—for 8 days. Loss of all teeth in upper and very many in lower jaw, some with, others without pain.	Convergent strabismus; marked weakness of right external rectus, both superior recti. In extreme position movements of eyeballs are accompanied by a tremor of eyebrows. Partial ptosis of both upper lids.	Atrophy of temporal and masseter muscles of left side. Sensibility: tactile: on forehead spots of anesthesia and delayed perception. Pain: analgesia over forehead, especially right; over both sides of the face large areas of hypalgesia. Temperature: on forehead, both sides, anesthesia; over face spots of anesthesia, delayed and perverted. Deep: disturbed on both upper lids. Sensibility: tactile: normal. Pain: areas of hypalgesia in center of forehead; over right side of face areas of hyperalgesia. Temperature and deep: normal.	Movement of right-side of face not so exaggerated as left, but on both sides somewhat ataxic.	Exercise improves considerably the ocular excursions.
No. 3—Denied.	Diplopia 2 years ago, lasted one year and disappeared completely.	Shooting pain in head. Lost 4 teeth since onset of disease, began to break down painlessly.	Some weakness of right externus. Both palpebral fissures diminished in size.			

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 4.—Venereal sore 18 years ago.	Never diplopia.	Loss of teeth in upper jaw.	Ocular movements show nystagmoid oscillations of the eye-ball on looking to right or left. Some weakness of the muscles, especially both external and internal recti. Partial ptosis of both eyelids—palpebral aperture much smaller.	Sensitivity: tactile: normal. Pain: normal. Temperature: normal. Deep: disturbed in the eyelids, normal in lips.	Normal.	Disease began with difficulty of vision and nystopia. As in case 1, the fact of early abolition of vision may have masked an eventual disorder of eye muscles.
No. 5.—Venereal sore 30 years ago.	Frequent and transient attacks of diplopia.	Formication of forehead, both eyelids, cheeks along both sides of the nose; suffered very frequently from hordeolum. Three years ago lost without pain all teeth in upper jaw except 4, dropped out while eating; in lower jaw all molars and bicuspid except one.	Convergent strabismus, marked weakness of both external recti, superior rectus left; during examination diplopia; partial ptosis of both upper lids; slight nystagmoid movements.	Some atrophy of left temporal. Sensitivity: tactile: normal. Pain: areas of hyperalgesia on both sides of forehead and nose. Temperature: disturbed. Deep: on upper eyelids slightly disturbed, lips more involved.	Marked over-winking of entire left side of face; marked winking of both upper lids in tight closure of eyes.	Test for deep sensibility somewhat difficult.

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 8 — Venereal sore 26 years ago.	Never diplopia.	None.	Ocular movements exaggerated—nystagmoid movements. Palpebral fissures less than normal and smaller on left than right.	Sensibility: tactile: normal. Pain: hypalgesia on forehead. Temperature: normal. Deep: normal.		
No. 9 — Venereal sore 20 years ago.	Never diplopia.	None.	Some nystagmoid movements on looking to right. Palpebral fissures smaller.	Sensibility: tactile: normal. Pain: hypalgesia on left side. Temperature: normal. Deep: somewhat disturbed on both eyes, especially left.		Double optic atrophy.
No. 10—Denied. Symmetrical suspicious looking scars.	Never diplopia.	Loss of teeth without pain.	Ocular movements normal. Palpebral aperture diminished.	Sensibility: tactile: normal. Pain: large areas of analgesia and hypalgesia on both sides of face. Temperature: anesthesia over lower portion of right face, areas of diminished and delayed sense. Deep: disturbed on right upper eyelid.	Normal.	

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Remarks.
	Eye- muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.	
No. 11—Venereal sore 23 years ago.	No diplopia.	None.	Ocular movements normal. Tremor of lids. Right pal- pebral aperture wider than left. Partial ptosis, es- pecially left.	Sensibility: tactile: normal. Pain: analgesia over left lower jaw laterally, hypalgesia over cheeks, etc. Temperature: left cheek same area anes- thesia, areas of hyperesthesia and hypesthesia. Deep: normal.	Facial In- ervation. Fairly good.
No. 12—Venereal sore 33 years ago.	Never diplopia.	Loss of teeth.	Ocular movements normal.	Sensibility: tactile: normal. Pain: both sides of face analgesia. Temperature: some- what disturbed on forehead. Deep: normal.	Right better than left; upon opening mouth wide left angle lower.
No. 13—Venereal sore 18 years ago.	Never diplopia.	None.	Ocular movements normal.	Sensibility: tactile: normal. Pain: area of hyp- algesia on central forehead. Temperature: nor- mal. Deep: normal.	Normal.

Syphilitic Infection.	Were there any symptoms of disease of eye-muscles?	Fifth nerve?	Present status.	Remarks.
No. 14—Venereal sore 12 years ago.	Never diplopia.	Since onset of disease suffers from hordeolum. Teeth excellent.	Eye-muscles. Ocular movements rather exaggerated, extreme positions are accompanied by tremor of eyebrows. Space between eyelids normal.	Facial In-nervation Both sides exaggerated.
No. 15—11 years ago.	Never diplopia.	Teeth decayed and fell out soon after onset of present disease.	Excursions of eyeballs exaggerated though eyes are quite prominent; some tremor of eyebrow on extreme fixation.	Sensibility; tactile: normal. Deep: normal. Temperature: normal. Deep: normal. Left forehead more wrinkled, mouth drawn more on left side and in opening mouth wide marked depression of left angle of mouth and left side of jaw.
No. 16—Denied.	No diplopia, no squint.	Years ago had neuralgia on both sides of forehead. One tooth in upper jaw, 7 in lower jaw.	Ocular movements normal.	Sensibility: all qualities: normal.

Syphilitic Infection.	Were there any symptoms of disease of		Present status.		Facial In-nervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 17—None.	No diplopia, no strabismus.	Loss of teeth.	Ocular movement normal. Narrowing palpebral aperture.	Sensitivity: tactile: normal. Pain: areas of analgesia and hypalgesia on both sides of face. Temperature: normal. Deep: normal.	Normal.	
No. 18—Denied.	No diplopia.	None.	Ocular movements normal. Tremor of eyelids.	Partial atrophy of right temporal. Sensitivity: tactile: Pain: normal. Temperature: hyp-peresthesia to cold on both sides, less so to heat. Deep: normal.	Normal.	Completely amaurotic.
No. 19—Denied.	No diplopia.	None.	Palpebral aperture narrow, especially on right eye.	Sensitivity: all qualities: normal.	Normal.	
No. 20—Denied One miscarriage.	Says that at times when feeling weak, sees double.	Some paresthesias of face. Loss of teeth since onset of disease.	Ocular movements good.	Sensitivity: tactile and pain: Normal. Temperature: large areas of anesthesia and perverted sensibility over both sides. Deep: normal.	Normal.	

Syphtic Infection.	Were there any symptoms of disease of		Present status.		Facial In-nervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 21.—Denied. One miscarriage.	Diplopia began 12 years ago and exists off and on ever since. Twitching of eye-muscles.	Very often neuralgic attacks, beginning in lower jaw like a tooth-ache; paresthesias of the forehead.	Ocular movements normal. Partial ptosis on both eyes.	Sensibility: tactile: normal. Pain: hyperalgesia on right cheek. Temperature: normal. Deep: normal.	Normal.	
			Excursions of eyeballs exaggerated; no evidences of paralysis, brings it out with ease beyond normal, some nystagmoid movements. Palpebral fissures unequal; semi-ptosis covers almost upper half of cornea, varies now and then; strabotic attitudes of eyes.	Evidences of slight atrophy of masticatory muscles. Sensibility: tactile: normal. Pain: delayed conduction, much analgesia — hyperalgesia — hyperalgesia chiefly on left side. Temperature: Deep: disturbed in eyelids.		
No. 22.—Denied.	Diplopia frequently.	Paresthesias of face. Loss of teeth without pain.			Ataxic.	

the deep sensibility of the facial and ocular musculature are not very exact. It is particularly difficult to ascertain slight disorder. The procedure described above gives a fair impression of the deep sensibility of the levator palpebræ, and no method is apparently known for determining the kinesthetic sensibility of the other external eye-muscles.

5. Some of the ocular palsies of tabes are probably sensorimotor palsies.

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201. LA LOCALISATION MOTRICE MÉDULAIRE (The Spinal Motor Localization.) Van Gehuchten and Nelis (Journal de Neurologie, Aug. 5, 1899, No. 16, p. 301).

The experiments of Marinesco, according to Van Gehuchten and Nelis, show that the peripheral nerves arising in the different plexuses do not have a definite nuclear localization in the spinal cord; the spinal motor localization is not "nervouse," *i. e.*, not determined by the nerves. Sano believed that each muscle of the body has a distinct nucleus in the gray substance of the spinal cord. The investigations of Sano, Kohnstamm and Marinesco show that the group of cells in connection with the diaphragm form a distinct column in the center of the anterior horn, but it does not follow that muscles of the extremities have a similar representation in the spinal cord. The motor localization is therefore not "muscular," but is "segmental," *i. e.*, the different groups of cells are in relation with muscles of a segment of a limb. Van Gehuchten and De Buck showed in an earlier paper that amputation of the leg at the knee caused chromatolysis in two groups of cells in the postero-lateral part of the anterior horn; one group extending from the upper part of the fifth lumbar segment to the lower part of the third sacral segment; the other from the upper part of the second sacral segment to the lower part of the fourth sacral segment. Van Gehuchten and Melis found in a case of amputation of both feet that the chromatolysis was limited to the lower group, from which they conclude that all the muscles of the foot are represented in this group, and those of the leg in the upper group. In the cervical cord of man we should expect to find three distinct cellular groups: one for the hand, one for the forearm, and one for the upper arm. We should find three groups also in the lumbosacral cord for the lower limb. It is not probable that these "segmental" nuclei contain "muscular" nuclei, *i. e.*, distinct cell groups for each muscle. The segmental division of the cells explains certain pathological processes: syringomyelia, progressive muscular atrophy, etc. SPILLER.